Information

Treatment of Shock Following Myocardial Infarction

JAY N. COHN, M.D.

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WHILE NEWER REFINEMENTS in patient monitoring and management have significantly reduced the mortality from acute myocardial infarction, the occurrence of shock still carries a grave prognosis. Once shock develops the survival of the patient is entirely dependent on the perception, attentiveness and judgment of his physician.

Shock is characterized by a critical reduction in tissue perfusion. Inadequacy of blood flow impairs organ function and disrupts the integrity of normal metabolic pathways. If shock is not promptly corrected, the flow deficiency leads to organ damage, metabolic acidosis and a vicious circle resulting in progressive circulatory deterioration and death. The sooner the syndrome can be recognized the more likely is therapy to be effective. The need for prompt recognition of shock must not, however, be satisfied at the expense of "over-diagnosis." It is in this initial evaluation that the physician's perceptiveness is critical. He must be able to recognize the difference between the mildly hypotensive patient who is adequately perfusing his tissues (and needs no immediate treatment) and the patient who is in the incipient stages of shock and requires prompt therapy to restore peripheral blood flow.

In considering the diagnosis of shock, attention should be given to the following signs:

Skin temperature. Warm skin indicates adequate cutaneous blood flow and usually a fairly well maintained cardiac output. Cool, clammy skin indicates sympathoadrenal discharge, a sign of reflex vasoconstriction in response to a fall in cardiac output.

Dr. Cohn is from the Hypertension and Clinical Hemodynamics Section, Veterans Administration Hospital, and the Department of Medicine, Georgetown University Medical Center, Washington, D.C.

Peripheral pulses. Thready or absent brachial and radial pulses indicate either severe hypotension or more often intense vasoconstriction. In either case urgent treatment is indicated. Femoral artery pulsation will be very weak if the patient is hypotensive but the pulsations are bounding in the presence of peripheral vasoconstriction.

Auscultatory blood pressure. This is not a reliable guide to intra-arterial pressure in shock. A low cuff pressure has the same significance as weak upper extremity pulses. However, an absent auscultatory pressure usually indicates inadequate blood flow and the need for treatment.

Mentation. If the patient is alert and responsive cerebral blood flow is probably adequate. Agitation, confusion or somnolence are signs of deficient cerebral blood flow and usually are associated with a fall in arterial pressure.

Urine output. Urine flow less than 20 ml per hour with a low urine sodium concentration is evidence of inadequate renal blood flow which if not corrected can lead to tubular necrosis.

Cardiac function. Persistent or recurrent chest pain or arrhythmias in the presence of other signs of hypotension may be accepted as presumptive evidence of functional impairment of coronary blood flow.

Acidosis. Low arterial blood pH and elevated blood lactate mean reduced tissue oxygenation. Arterial blood gas and pH studies are invaluable in the management of patients in shock.

The presence of one or more of the above signs of inadequate tissue blood flow in a patient with an acute myocardial infarction is presumptive evidence of shock. Mild hypotension in the absence of any of these signs should not be diagnosed or treated as "shock."

When the diagnosis of shock has been made, several questions regarding the hemodynamic status of the patient should be answered before definitive treatment can be instituted:

Is the patient severely hypotensive? Hypotension is an immediate threat to life because of the associated impairment in cerebral and coronary blood flow. Since the cuff pressure may be low even though arterial pressure is normal, the strength of femoral arterial pulsation often is a more reliable guide to blood pressure. In some patients direct recording of arterial pressure may be necessary.

Is blood volume adequate? Some patients become hypovolemic in the hours following an acute myocardial infarction and the reduction in plasma volume may then become an important factor in the genesis of shock. The central venous pressure (CVP) is a vital guide to the adequacy of circulating volume and should be monitored in all patients with shock. This can be accomplished by threading a catheter through a needle in the brachial, femoral or subclavian vein and advancing it into the thorax. A low CVP (less than 6 cm of water with the zero level at the mid-chest) is an indication for a trial of volume expansion. In myocardial infarction the left ventricle often is in failure while CVP is normal. Therefore, volume expansion should be carried out cautiously. A rise in CVP of more than 2 cm of water during infusion of dextran, saline or other fluid indicates that volume has been adequately restored. If shock is not corrected by volume expansion the presence of significant left ventricular failure can be assumed.

Is cardiac function severely impaired? If peripheral blood flow is decidedly reduced and the CVP is high, then myocardial failure is obviously an important factor in the shock. Heart rate is not a very useful index of cardiac function. Indicator dilution cardiac output data are of value in the evaluation of myocardial function in selected cases.

What is the status of the peripheral vessels? Is there evidence of intense sympathetic discharge? This usually is manifested by cutaneous vasoconstriction and indicates renal vasoconstriction as well. In early stages of shock peripheral constriction may support fairly normal arterial pressure despite progressive tissue hypoperfusion and lactic acidosis.

The purpose of therapy in shock is to restore adequate organ perfusion. Effective therapy must be based not only on an understanding of the physiological disturbance in the individual patient but also on a thorough understanding of the pharmacological action of the useful drugs.

The following drugs may be valuable in certain patients with cardiogenic shock:

Isoproterenol. This is a catecholamine with pure beta adrenergic activity; that is, it stimulates the heart and dilates peripheral vessels. It is probably the agent of choice when impairment of cardiac function has led to severe reduction in cardiac output, especially when reflex vasoconstriction is present. Isoproterenol 1 or 2 mg should be diluted in 500 ml 5 percent dextrose in water and the rate of infusion gradually increased until the signs of shock are corrected or cardiac rhythm disturbance limits further administration. In some cases the concentration of isoproterenol must be increased as much as 2 mg per 100 ml to obtain a satisfactory effect. Lidocaine may be effective in controlling ventricular irritability during isoproterenol infusion. In some hypotensive patients isoproterenol will not significantly increase arterial pressure and cerebral and coronary perfusion are not improved. In this situation a vasoconstrictor-inotropic agent may be necessary.

Levarterenal (Norepinephrine) or metaraminal. These drugs have an alpha adrenergic effect (vasoconstrictor) on peripheral vessels combined with myocardial stimulating properties. Because these drugs may reduce renal and splanchnic blood flow they should be used only when isoproterenol is ineffective. The infusion rate should be the smallest amount necessary to increase systolic arterial pressure over 100 mm of mercury.

Digitalis. The cardiac glycosides have inotropic effects less potent than the catecholamines. They also have vasoconstrictive properties when used intravenously. It is probably best to treat cardiogenic shock acutely with the adrenergic inotropic drugs above and to administer digitalis orally for its more sustained effect.

Atropine. If shock is associated with sinus bradycardia, 1 mg atropine intravenously may be effective in restoring heart rate and blood flow. Drugs, such as atropine and isoproterenol, which result in an increase in atrial rate must be used cautiously in the presence of atrioventricular block. In these circumstances, an increase in atrial rate may result in a decrease in ventricular rate.

Furosemide. This potent diuretic can help establish urine output in the oliguric patient. After shock has been treated with the vasoactive compounds above a diuretic response to intravenous infusion of 200 mg of furosemide indicates that renal perfusion is adequate. If oliguria persists, however, more aggressive attempts to improve blood flow are necsesary.

Sodium Bicarbonate. If the arterial pH is less than 7.35, sodium bicarbonate should be administered in amounts adequate to restore pH to above that level. Treatment should be initiated with 40-100 meq sodium bicarbonate and further alkali therapy based on arterial blood pH measurements.

Ventricular pacing. If shock and marked bradycardia co-exist, increase in ventricular rate via catheter electrode pacing is often of great clinical benefit.

Newer pharmacological approaches such as the use of sympathetic blocking agents and other inotropic drugs, such as dopamine and glucagon, are still in the experimental stage.

Effective management of shock requires not only initiation of the correct therapy in the correct amounts, but also close continuous monitoring of cardiovascular function. Adrenergic drugs should be weaned and discontinued as soon as possible. Blood volume may be inadequate after cardiac function is improved, and a falling CVP may be an indication for administration of dextran, even in patients who have manifested heart failure only a few hours before. If rhythm disturbances persist electrical pacing through a transvenous pacemaker may help improve peripheral blood flow.

It is clear that intelligent use of the means currently available can be effective in salvaging many patients who would otherwise succumb to cardiogenic shock. In others, however, the impairment in cardiac performance is so severe that medical therapy is ineffective. In this selected group of patients mechanical means of temporary circulatory support may eventually become an important adjunct to management.

Pesticide Poisoning May Appear Anywhere

A Statement prepared by the Committee on Occupational Health of the California Medical Association in cooperation with the Bureau of Occupational Health, California Department of Public Health.

CONTAMINATION OF CONSUMER goods from spills of toxic chemicals occurring in storage and transit have resulted in bizarre and tragic episodes of poisoning involving hundreds of people. Incidents of less severe nature, but somewhat similar to those that have occurred in Colombia, Saudi Arabia and Mexico, have taken place in California. The increasing amount of such materials being transported and stored in California increases the probability of further accidents here. Significant contamination can occur from a very small amount of a very toxic pesticide and a poisoning may result from either ingestion or skin absorption. There has been an increase in spills of toxic pesticides due to containers falling from trucks on the highways. There has also been an increase in the use of such pesticides for suicidal purposes.

Every physician in California, whether practicing in an urban or a rural area, should be able to recognize and treat promptly poisoning from phosphate ester pesticides. This capability can frequently save lives of poison victims who have absorbed several potentially fatal doses.

The antidotes for poisoning by these anticholinesterase chemicals are large doses of atropine and Protopam chloride.* An adequate supply should be readily available to the physicians and hospitals in all areas of the state.

There are hundreds of pesticides ranging in wide degrees of toxicity. The name or type of chemical must be known before specific treatment can be instituted. However, it is the phosphate ester pesticides with which the physician should be most familiar. Among the most toxic are TEPP (tetraethylpyrophosphate), Phosdrin® (alpha isomer of 2-carbomethoxy-1-methylvinyl dimethyl phosphate), parathion, methyl parathion, Thimet® (phorate), Di-Syston® (sulfur analog of demeton), and Systox® (demeton).

Diagnosis and Treatment

Signs and symptoms are explainable on the basis of cholinesterase inhibition. Symptoms may be delayed for several hours after last exposure, but rarely for a longer period than 12 hours. Early or mild poisoning is hard to identify since it can be confused with other conditions, such as heat exhaustion, gastritis, encephalitis, asthma, pneumonia, or other respiratory infections. Glycosuria can be found in 30 percent of the cases and diabetic coma mistakenly considered. Symptoms most often appear in the following order: headache, fatigue, giddiness, nausea, salivation, sweating, blurred vision, tightness in chest, abdominal cramps, vomiting, and diarrhea. In severe poisoning, difficult breathing, tremors, convulsions, collapse, coma, pulmonary edema, and respiratory

*Protopam chloride® (pralidoxime chloride, 2-PAM) is a product of the Ayerst Laboratories, Inc., New York, N.Y.